ETIO PATHOGENESIS AND MANAGEMENT OF

ILEAL PERFORATION

DISSERTATION SUBMITTED FOR

BRANCH - I M.S (GENERAL SURGERY)

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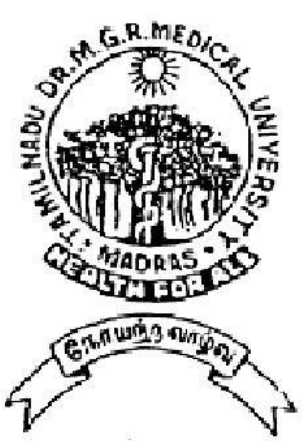

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CERTIFICATE

This is to certify that the dissertation entitled **"ETIO PATHOGENESIS AND MANAGEMENT OF ILEAL PERFORATION"** is the bonafide work of **Dr. LAKSHMANA.R.** in partial fulfillment of the university regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for M.S (Branch I) General Surgery examination to be held in April 2013.

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DECLARATION

I, **Dr. LAKSHMANA.R.** hereby declare that, I carried out this work on "**ETIO PATHOGENESIS AND MANAGEMENT OF ILEAL PERFORATION"** at the department of surgery, Govt. Rajaji hospital, Madurai, under the guidance of Prof. Dr. S. Selva chidambaram, M.S., PROFESSOR OF SURGERY, during the period of June 2011 to June 2012. I also declare that this bonafide work has not been submitted in part or full by me or any others for any award, degree or diploma to any other university or board either in India or abroad.

This is submitted to the Tamilnadu DR. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S degree examination in general surgery (branch I) to be held in April 2013.

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CONTENTS

INTRODUCTION

Ileal perforation is a common problem seen in tropical countries. The commonest cause being typhoid fever. In western countries the causes are malignancy, trauma and mechanical etiology, in the order of frequency.

Over the years a definite changing trend has been observed in ileal perforations both in terms of causes, treatment and prognosis. Better antibiotics, aggressive surgery and the elimination of conservative treatment, better preoperative and postoperative care have all significantly contributed to the improvement in patient outcome.

It is true that outcomes have improved but still cases of ileal perforation cause a significant morbidity and mortality that persists despite the significant changes in health care over the years

This dissertation aims to study the etiology, presentation, management outcome and the factors influencing prognosis and outcome in ileal perforations

The present study includes 40 patients of ileal perforation with emphasis on typhoid, and nonspecific perforations and the factors influencing outcome.

AIM AND OBJECTIVES OF THE STUDY

The aims and objectives of this study are

- To study the presentation and management of patients admitted with ileal perforation
- To study the outcome of these patients
- To study the factors influencing the outcome in these patients

HISTORY

In the Sushruta Samhita, intestinal perforation by sharp objects such as a fish bone or a thorn has been described. It was also recorded that the abdomen was opened and the bowels drawn out. If the intestine was severed or perforated the edges were held together and large black ants allowed to clamp the cut ends with their jaws, prior to their bodies being clipped off. The gut was reintroduced into the abdominal cavity and the incision closed[6], The text also states that is the intestines were dirty, they were to be rinsed, and then washed with milk and clarified butter [6] William Cullen coined the term 'peritonitis' in 1776 Benjamin Travers did the first successful closure of an intestinal perforation[8] The introduction of Lempert's sutures was a significant advancement in the technique of restoring intestinal continuity [7]

Hippocrates first used the term typhus (gr. cloudy) in 460 B.C [9] In 1829 Louis used the term typhoidae and described 150 cases with intestinal perforation, haemorrhage, splenomegaly, rose spots and mesenteric lymphadenopathy. Budd in 1973 proposed that the disease spreads through water contaminated with excreta.

Karl Joseph Ebereth discovered the Typhoid bacillus in 1880. In 1884, Gaffkey first isolated and cultured Salmonella typhi [10] Widal described the test to detect agglutinins in the serum of patients suffering from typhoid fever in 1896. The first vaccine for human use against typhoid was made by Pfeiffer and Kalle in 1896 [10].

Etiology

The causes of ileal perforation described in literature are

1. Typhoid
2. Trauma
3. Tuberculosis
4. Mechanical Causes
5. Malignancy
6. Ischemic Enteritis
7. Crohn's Disease
8. Non Specific
9. Perforation due to Diverticula
10. Miscellaneous

In a series published by Karmakar et al, the causes of ileal perforation were as follows [1]

Review Table 1

Causes	Number
Typhoid	17
Tuberculosis	1
Round Worm	2
Meckel's	1
Blunt Trauma	1
Penetrating Injury	1
Non Specific	7
Total	30

As shown in Review Table 1, typhoid fever was the most common cause of ileal perforation in Karmarkar's series, followed by nonspecific perforation. There were no cases of perforation secondary to malignancy [1]

In other studies the causes have been as follows [2,3,11]

Review Table 2

Author	Year	country	Cause	Number
Bhalerao[11]	1981	India	Nonspecific	18
			Typhoid	8
			Tuberculosis	3
			Trauma	8
			Diverticulitis	2
			Total	32

In the series by Bhalerao in India, non-specific and typhoid fever were the commonest causes [(11)]. The commonest cause of ileal perforation in the tropics is typhoid fever [(1,9,10)]. In the western population the causes are described to be mechanical causes, malignancy and trauma [(2,3,12,13)].

Typhoid Perforation

Incidence

The reported rate of bowel perforation iii typhoid fever varies from 0.5% to 78.6%. Various studies have shown the following incidences

Review Table 3

Author	Year	Country	Number
Purohit	1976	India	0.5%
Archampong	1976	Ghana	20.5%
Thakkar	1979	India	3.77%
Arigbabu	1980	Nigeria	78.6%
Hadley	1984	South Africa	4%
Santillana	1991	Peru	7.8%
Hisconmez	1992	Turkey	0.58%

Santillana calculated an incidence of 5% in a collective series [18]

Sex and Age

There is a male preponderance in typhoid perforation. It predominantly occurs in younger age groups. It has been reported in patients from age 2 to 76 years [5,16,18,21,22,23,24,25] .Perforation predominantly occurs in the second and third decades of life [4,5,26] .The age and sex incidence of perforation is given in Table 4.

Review Table 4

Author	Year	Male: Female Ratio	Age (years)	Mean Age
Thakkar	1979	5.2: 1	6-54	-
Eggeiston	1979	3.1: 1	4-65	28
Tarpley	1989	2.3: 1	-	27.1
Santillana	1991	6: 1	4-56	-
Mock	1992	2.4: 1	2-72	16.7
Tacyildiz	1995	2.4: 1	3-70	27
Singh	1995	3: 1	-	14.2

Seasonal Variation

Eggeiston reported that over half the cases occurred between July and October [26] Hadley reported that 58% of cases occurred in the dry season [17] Tarpley reported that 27% of patients were admitted during the driest quarter of the year and 21% during the rainiest quarter [23]

Pathology

Typhoid fever is caused by a Gram-negative bacillus Salmonella typhi. The organism passes through the Peyer's patches without causing inflammation. Multiplication occurs in the reticuloendothelial system for 10-14 days. Seeding occurs into the blood stream corresponding to the clinical onset. During the second week of illness bacteria reach the gut and localize in Peyer's patches. Ulceration and mesenteric adenitis occurs. Necrotic areas appear in lymphoid tissue. This might lead to perforation of Peyer's patches [18,27,28] .Perforation is reported to occur commonly in the second week following onset of illness [17,18,24,25] .Keenan reported that 88% of patients perforated in the second week [17].

Santillana reported a patient who perforated within 24 hours of onset of clinical illness[18] .The timing of perforation in a series of 59 children reported by Lizzaralde is as follows [25]

Review Table 5

Timing	N	%
First week	8	13.5%
Second week	32	54.2%
Third week	13	22%
Fourth week	6	10%

Macroscopy

Peyer's patches are swollen and raised. Mesenteric nodes are enlarged. The terminal ileum and caecum are affected. Ulceration occurs in the long axis of the bowel. Perforation diameter varies with a mean of 5mm. Hadley reported that most of the perforations are smaller than 5mm [17]. Tarpley noted that the size of the perforation varied between 1mm and 6cm in size with most being less than 8mm in size [23] .Multiple perforations are seen in 20% of patients. Hadley

reported multiple perforations in 17% of patients varying from two to six. Nine patients had caecal perforations[17].Santillana reported two perforations in 16% of patients and more than two perforations in 6% of patients [18] .Mock et al reported the following in their series [21].

Review Table 6

Single Perforation	78.5%
Two Perforations	13.3%
Three Perforations	4.1%
Four Perforations	4.1%

Meier reported multiple perforations in 19% of patients with the maximum up to 10 and average being 1.4 [23] .Most of the perforations occur within 30cm of ileocaecal junction[17,18].

Microscopy

There is marked proliferation of reticuloendothelial cells of the lymphoid follicles locally and systemically. There is accumulation of mononuclear phagocytes. The macrophages form small nodular

aggregates filled with red cells (erythrophagocytosis). The bacteria are sometimes visualized [29]

Clinical Features

Typhoid fever is characterized by fever, splenomegaly and Rose spots. Patients have headache, respiratory complications and relative bradycardia. Complications include myocarditis, pneumonia, gastrointestinal bleed, cholecystitis, osteomyelitis and intestinal perforation [28,30] .The onset of perforation is heralded by sudden increase of abdominal pain, vomiting and distention. However these signs may be obscured in a patient who in a toxic state is obtunded resulting in a delay in diagnosis and treatment [18].

Meier et al reported the following symptoms and signs [23].

Review Table 7

Symptoms

Fever	93%
Abdominal Distention	73%
Abdominal Pain	90%
Rectal Tenderness	24%
Vomiting	67%
Diarrhoea	27%
Constipation	24%

Eggleston reported that most of the patients had fever, malaise and sudden increase in abdominal pain. Examination revealed signs of toxemia and acute abdomen. Hyper resonance was present over the liver in 70% of patients and paralytic ileus in 68% of patients. 19.2% of patients were in shock [(26)]

Diagnosis

Clinical suspicion is often sufficient for diagnosis in endemic area[(17,18,19,31)] Free gas may be present under the diaphragm. Pneumoperitoneum has been reported in 52% to 82% of patients

[17,19,22] Abdominal paracentesis may reveal pus. Bhalerao et al reported positive suprapubic aspiration in all 32 patients with small bowel perforation. Peritoneal lavage might be useful to detect bile or pus[11].

The diagnosis of typhoid fever can be made by Widal test, culture of organism from blood, bone marrow, urine and stools. Newer diagnostic techniques have been introduced to enable rapid diagnosis of typhoid fever. Histopathology of the specimen might reveal etiology of perforation.

Serology

Widal test measures antibodies against the flagellar and capsular antigens of the causative organism. A positive diagnosis can be made from seventh to tenth day. This test is of less value in low endemic regions. Widal test was reported positive in 46.1% of tested patients by Santillana [16,18]. Kaul obtained a positivity rate of only 30% [32] .Rising titres might be more useful[33] .Four-fold rise in titres is considered diagnostic but the rise may be blunted by early antimicrobial therapy [38] .Agglutinins might be present on account of prior disease. H agglutinins persist longer than 0 agglutinins.

Persons who had prior infection may develop anamnestic response during an unrelated fever but the rise is transient [35].

Culture of Organism

Salmonella typhi can be isolated from blood, bone marrow aspiration stool and urine. In untreated patents blood culture is positive in 80% of the patients in the first week declining to 20-30% during later stages. A success rate of 90% is obtained from bone marrow aspirate culture. Prior treatment makes culture less likely [33].Hadley reported positive blood culture in 22.2% of tested patients [17]. Santillana reported positive blood culture in 48% of patients [18] .Bone marrow might yield Salmonella typhi in the absence of a positive blood culture [33].

Stool cultures are frequently negative during the first week but are positive in 75% by the third week. The frequency of positive urine culture parallels that of stool culture and in some cases may represent fecal contamination [34]. Other sources of positive cultures may be duodenal aspirate, rose spots, pus from suppurative lesions, CSF and sputum but are not of practica1 value [35].

Histopathology

The presence of macrophages and erythrophagocytosis virtually marks the diagnosis of typhoid perforation. Organisms may sometimes be demonstrated [29].

Newer Methods

Currently several newer methods of diagnosis are under evaluation. Indirect hemaggluination, indirect fluorescent Vi Antibody and ELISA are more specific and sensitive when compared to the Widal test [33] .The use of monoclonal antibodies against Salmonella typhi flagellin and DNA probes for detection of Salmonella typhi in blood are promising developments. The newer techniques would enable rapid detection of antibodies or organism[33].

Treatment

Appropriate management of typhoid perforation was controversial till 1960. Finney and Cushing advocated surgical treatment but with the advent of chloramphenicol some authors started recommending conservative management. Huckstep advocated conservative treatment in 1959. He proposed management

of typhoid perforation on the lines of the Oschner-Scherren regimen. His reasons for this were:

- The terminal ileum is friable and is liable to perforate at more than one spot. The friable gut might not hold sutures.

- Chloramphenicol therapy sterilizes bowel contents and adjacent loops might localize the perforation [36].

Hook and Guerrant recommended surgery if there was no localization [37]. A standard text had earlier stated that the results of surgery were so poor that it should not be advocated but a recent edition has advised surgical management[38,39].

Conservative management is associated with a substantial mortality. Arigbabu and Badejo reported 66% mortality with this modality[20]. Presently all authors recommend surgical management[20].

Surgical Treatment

Patients are resuscitated preoperatively with intravenous fluids and antibiotics. Tacyildiz et al reported that preoperative resuscitation, antibiotic therapy and total parenteral nutrition reduced mortality from 28.5% to 10%[20]. The various surgical options are

1. DRAINAGE OF PERITONEAL CAVITY

It is done in moribund patients during resuscitation and preparation for surgery [40]. Flank drains are inserted under local anesthesia. As the only procedure it carries an unacceptably high mortality. It may be used as a temporary measure or as a preliminary step prior to surgery in moribund patients.

2. SIMPLE CLOSURE

Freshening of the edges and closure has been recommended by Archampong[31] .He reported mortality of 17.3% with this procedure. Talwar et .al recommended primary closure and limited surgery [41]

3. WEDGE RESECTION AND CLOSURE

A wedge of ileal tissue is resected around the perforation and the defect is closed transversely in two layers. Mortality rates between 2.3% to 6.2% have been reported [18,42] .Ameh reported that a wedge resection is associated with a very high mortality rate[42].

4. RESECTION - ANASTOMOSIS

Excision of the affected segment and anastomosis has been recommended by some authors. Ameh et al recommended resection anastomosis over wedge resection and simple closure [42]. Jarrett and Gibney recommend resection only for multiple perforations. Gibney recommended resection if there were three or more perforations [28, 40]. Other earlier studies showed varying mortality between 7.6% and 67% [18,26].

5. ILEOTRANSVERSE ANASTOMOSIS

Simple closure, wedge resection or a resection anastomosis may be combined with a side-to-side ileotransverse anastomosis. Closure of the terminal ileum and end to side ileotransverse anastomosis was recommended by Eggleston. Higher incidence of

complications was seen in patients treated by simple closure when compared to ileotransverse anastomosis [43]. Lizzaralde recommended ileotransverse anastomosis to decrease complications. Ileotransverse anastomosis helps by diverting the faecal stream from diseased ileum and decreases the risk of complications [25].

6. ILEOSTOMY

Lateral tube ileostomy is recommended by some authors. Kaul treated 9 patients by placing a Foley's catheter through the perforation with two deaths [32]. Lizzaralde recommends simple closure of perforation and ileostomy through normal bowel. Lateral ileostomy may be used where the bowel is relatively friable and it helps decrease the intraluminal pressure [25].

Bhalerao et al recommended exteriorization of suture line, which prevents contamination of the peritoneal cavity in case of leak. Santillana recommended exteriorization in moribund patients. If fistulae form they invariably heal on conservative management [10,18]. Good peritoneal lavage and placement of drains to remove pus was recommended. Two-layer closure was recommended to

decrease the risk of leakage [21,24]. A midline or Para median incision was commonly used. Talwar et al recommended Rutherford Morrison incision in the presence of a confirmed preoperative diagnosis of perforation [45] .If there is fulminant sepsis in the abdominal cavity due to the formation of faecal fistula or any other cause laparostomy might be done. Laparostomy is defined as a laparotomy without reapproximation and suture closure of abdominal fascia and skin. The abdominal cavity is left open. It helps drainage of pus and prevents deleterious rise of intra-abdominal pressure. The wound can be closed after control of sepsis. The disadvantages are that the exposed intestine might perforate and formation of an incisional hernia. It may be combined with continuous postoperative peritoneal lavage[45].

Medical Therapy

Chloramphenicol was the gold standard for the treatment of typhoid fever. The recommended dose is 3-4g/day or 50-70mg/kg for children. The dose may be slowly reduced to 2/day or 30mg/kg once the patient is afebrile. Rapid response occurs within 24-48 hours. The duration of treatment is 2 weeks. Other drugs that have been used are amoxicillin and co-trimoxazole[46].

With the advent of resistance to this drug, quinolones have replaced chioramphenicol as the drugs of choice [47,48]. Ciprofloxacin is used in a dose of 200 to 750 mg twice a day. Resistance to this drug is still rare [47]. Cefiriaxone may be used as an alternative. The dose is 3-4g/day for 3 days in adults and 80mg/kg/day in two divided doses for 5 days in children [47].

In the event of bowel perforation with peritonitis, gentamicin and metronidazole should be added to the anti-typhoid drugs [47]. Vaidyanathan reported that metronidazole when added to the preexisting anti-typhoid drugs greatly reduced mortality [49].

Complications

Typhoid perforation is associated with a very high morbidity. Complication rates of 28.5% to 81% have been reported [17,18,22,24,26] Santillana in his series of 96 patients reported a complication rate of 71.9%[18] The complications and their rates seen in two series are shown in table 8. The common complications are wound infection, wound dehiscence and respiratory complications [17,18,21,23]. Intestinal fistulae have been reported in 3-10% of patients [17,18,21] .Santillana and Meier reported a fistula rate of 3% [18,23] .Hadley reported a rate of 10% [17]. Ihkwaba and Shittu favoured early closure of intestinal fistula in developing countries where resources are limited [49]. Reperforation has been reported in 1-10% of patients [17,18,21,23]. Santillana reported reperforation rate of 1.04% [18] .Hadley reported a reperforation rate of 10% [17].

Review Table 8

Author	Year	Complications
Keenan and Hadley	1981	Chest Infection Septicemia Wound Infection Reperforation Recurrent typhoid GI Fistula Abdominal Wall Fascitis Wound dehiscence
Santillana	1991	Wound Infection Chest Infection Renal Failure GI Fistula Melena Icterus Septicemia Reperforation Incisional Hernia Pleural Effusion Parotid Abscess *

Mortality

Typhoid perforation is associated with a significant high mortality rate. This is increased in case patients are managed conservatively.

Mortality rates of between 3 and 60% have been reported in patients managed surgically. The rates are as shown below.

The causes of mortality in a series of 68 patients reported by Archampong are follows [19]

Review Table 09

Cause of Mortality	Percentage (%)
Toxemia with myocarditis	45.6
Shock and dehydration	23.5
Aspiration Pneumonia	13.2
Bronchopneumonia	7.4
Renal Failure	5.9
Confusional State	4.4

Prognostic Factors

A variety of factors are known to influence survival including late presentation, female sex, age over 40 and multiple perforations[28]. Archampong reported that the duration of illness, perforation-operation interval, urinary output before surgery, blood urea and serum potassium influenced survival. Survival was independent of hemoglobin level, presence of peripheral circulatory failure, sickling status and number of perforations[19]. Relationship between perforation-operation interval and mortality as shown by Archampong is shown below [19].

Review Table 10

Time Interval	Mortality (%)
<24 hours	14.1
24-48 hours	22.8
49-72 hours	31.3
>4 days	80

Mock et al reported that increasing number of perforations, generalized contamination of the peritoneal cavity and single layer closure increased the mortality [21]. The relationship between the number of perforations and mortality as reported by Mock is as follows

Review Table 11

Perforations	Mortality %
1	27
2	31
3	50
>3	80

Eggelston and Santoshi in their experience with 78 patients reported that duration of illness, duration of perforation prior to surgery, presence of shock, uremia, encephalopathy, faecal peritonitis and postoperative fistula significantly influenced survival. Mortality was independent of procedure done [26].

Review Table 12

Weeks	Cases	Deaths	Mortality %
<1	15	1	7
1-2	25	8	32
2-3	17	9	53
3-4	4	1	25
>4	17	6	35

Bose et al reported that mortality in small bowel perforation was significantly influenced by perforation-operation interval, presence of multi-organ system failure and septic shock. Mortality was no influenced by haemoglobin, serum electrolyte levels age and sex of the patients. Patients were stratified in to four groups depending on their general condition.

Group 1 — Patients with normal parameters

Group 2 — Patient is conscious, afebrile, PR 90-11 0/mm, SBP 90-110mm Hg, Urine output> 30m1/hour

Group 3 — Patient is febrile, moderately dehydrated with PR 110-1 30/mm, BP 80-90 mm Hg, Urine output 20-3Oml/hour

Group 4 — Patient is disoriented, BP < 80 mm Hg, febrile or hypothermic, Urine output < 20m1/hour

There was no mortality in the first two groups whereas groups 3 and 4 had a mortality of 19.29% and 53.8%, respectively [50].

Talwar and Sharma reported that increasing the time interval between the perforation and surgery and feculent peritonitis increase the mortality. Mortality was least with early primary closure [41]

Some studies have found mortality to be associated with the type of surgical procedure performed. Ameh reported 50% mortality with simple closure, 62% with wedge resection and 36% with resection and anastomosis [42] Eggleston, though, did not find any such correlation.

Review Table 13

Author	Year	Drainage	Simple Closure	Wedge Resection	Resection	Ileostomy	Ileo traverse
Janet	1975	20	10.2	-	7	-	10
Eggleston	1979	33	30	-	67	-	31
Santillana	1991	10	-	2.3	7.6	50	-
Ameh	1997	-	50	62	36	-	1

Early prognostic evaluation of abdominal sepsis can easily be done by various scoring systems. Acute Physiology and Chronic Health Evaluation score (APACHE II)

Manheim peritonitis index predict the outcome of peritonitis. The latter is easier to apply for prognostication and is shown in table14

Review Table 14: Mannheim peritonitis index.

Risk factor	Weightage
Age>50 years	5
Female sex	5
Organ failure*	7
Malignancy	4
Pre-operative duration of peritonitis >24hrs	4
Origin of sepsis not colonic	4
Diffuse generalized peritonitis	6
Exudates	4
Clear	0
Cloudy, purulent	6
Faecal	12

Definitions of organ failure

Kidney	Creatinine level ?l77mmol/l
	Urea level >1 67mmolIl
	Oliguria < 20m1/h
Lung	P02 < 50mmHg
	PCO2 > 50mmHg
Intestinal obstruction	Paralysis 24 hr. or
	complete mechanical ileus

Malignancy

Small intestinal malignancies are very rare accounting for 1-3% of all gastrointestinal malignancies. The reported small bowel tumors in order of frequency are adenocarcinoma, carcinoid, lymphoma and sarcoma. The commonest site is the Ileum. Lymphomas are the commonest small bowel tumors to perforate. Dixon et al in their series of 54 cases had 9 cases of lymphoma and two perforations due to small bowel carcinoma [(2)].Rajagopalan and Pickleman in their series of 16 patients with free perforation of the small intestine had 2 patients with lymphoma[(13)]. Lymphomas often involve the bowel wall centrifugally leading to perforation. This may occur in an area of cancerous involvement often secondary to partial or complete distal obstruction[(13),]Resection of the segment and the adjacent mesentry is recommended[(61)],

Non-specific Perforation

When the etiology of ileal perforation is not identified, it is termed as a non-specific perforation. Dixon et al in their series had such results in 14 out of 54 patients [(2)]. Karmakar et al in their series of 30 patients of ileal perforation had 7 cases of non specific

perforation [1]. Many of these cases may be due to undiagnosed typhoid or other non-specific causes such as diet, drugs, viral or parasitic infections and infestations. It was earlier attributed to undiagnosed typhoid but these patients have different outcomes when compared to those with typhoid perforation. It has been proposed that sub mucosal vascular emboli may be responsible for such perforations [64] Drugs such as potassium tables may cause ulceration and subsequent small bowel perforation [65].

Diverticulitis

Perforation of diverticula is a rare cause of small bowel perforation. Huttunen et al in their series of 24 patients of perforation had this as the etiological factor in 4 patients, one with perforated ileal diverticulum, two with diverticulitis and one with ectopic gastric mucosa in a perforated Meckel' s diverticulum [66]. Bhalerao et al had two patients with perforated diverticula in their series of 32 patients [11].

Meckel's diverticulum occurs in 0.3% to 2.5% of population. Gastric mucosa is found in up to 38% of Meckel' s diverticula [67]. Perforation of an acquired diverticulum is rare. The gastric mucosa in a Meckel's diverticulum may lead to ulceration, which might perforate [66]. Resection of the diverticulum with the adjacent ileum is recommended[12,67].

Ischemic Enteritis

Ischemic enteritis is a rare cause of ileal perforation. Dixon in his series of 54 cases had 3 due to this cause [2] The gross lesion can be described in four stages

1. Segmental bluish discoloration, edema and mucosal ulceration
2. Circular purple bands with edema of bowel wall
3. Intestinal segment becomes longer, rigid and pipe-like
4. The segment becomes thin and papery

Perforation usually occurs in the fourth stage. Histological picture s severity of which varies with the stage of the disease [68].

Miscellaneous

The miscellaneous causes reported are roundworm infestations, pc radiation enteritis, steroid dependency, and AIDS[1,2,3]. Remine reported 79 patients on steroids at the time of

perforation. Patients receiving Prednisolone at 20mg/day had a mortality of 85.1%. Medical problems necessitating steroid theraphy were myeloproliferative disorders, connective tissue disorders and metastatic patients. The risk of perforation was highest during the first three steroid theraphy [(69)].

Sunke et al reported a patient of AIDS with an ileal perforation infection was postulated as the cause in this case[(70)].

Radiation can lead to perform due to impairment of blood flow and mucosal inflammation[(66)].

Tuberculosis

Tuberculosis is frequently an under diagnosed cause of ileal perforation. Agarwal and Gera reported tuberculosis as the etiology in 9 out of 113 specimens of ileum examined[57] Karmakar et al reported one case of tuberculosis perforation in their series of 30 cases of ileal perforation[(1)]. Bowel is involved by ingestion of infected saliva or sputum following pulmonary tuberculosis. Perforation commonly occurs in the hypertrophic variety proximal to the stricture[(58)] Wig et al reported that all their ten patients perforated through the ulcer. The incidence of perforation in tuberculous enteritis has been reported to be between 1 and 10%. Multiple strictures and perforations may also be seen. Kakar found multiple perforations in 36.3% of cases and strictures in 72.6%. The diagnosis of tuberculosis is made by demonstrating the tubercles in the intestine and mesenteric lymph nodes. Wig et al could show acid-fast bacilli in 40% of resected specimens. Resection-anastomosis is the procedure of choice. An alternate procedure is resection of the involved segment with ileo-transverse anastomosis. Simple repair is the only procedure possible in some poor risk patients but is associated with increased risk of leak and fistula formation [(58,59)]

High mortality rates between 30- 60% have been reported. Kakar reported a higher mortality in patients with multiple strictures and perforations [58]

Mechanical Causes

When the perforation occurs secondary to a distal obstruction due to causes such as hernias, bands, volvulus, intussusception and obstructing growths it is considered to be due to a mechanical cause. The cause is vascular strangulation following obstruction either by a hernia or a band. And gangrenous segment of bowel ruptures possibly as a result of delayed surgical treatment[27]. Increased intraluminal pressure may also lead to perforation. Mechanical causes are the one of the commonest causes of bowel perforation in the western world. These were responsible for 18 out of 76 cases of small bowel perforation as reported by Chaikof. The causes were adhesions in 12 patients, hernia in 4 and obstructive carcinomas in 2 patients . Dixon et al in their series of 54 patients reported 13 cases due to mechanical causes - adhesions in 8, colonic cancer in 2, gall stones in 2 and small bowel volvulus in one patient.

Inflammatory Bowel Disease

Free perforation is a rare complication in Crohn's disease. Ileum is the commonest site of perforation in this disease. Steinberg et al in their series of seven patients of Crobn's with free perforation of small bowel had five with ileal perforation. Dixon et al in their series of 54 patients had 5 with Crohn's disease. Chaikof reported 16 cases of Crohn's in the 76 patients of non-traumatic small bowel perforation. Perforation in Crohn's disease occurs during an acute exacerbation and is usually associated with distal obstruction. Simple closure is inadequate and has poor results. Menguy recommends primary excision and creation of a double-barreled ileocolostomy with closure of stoma at a later date.

ANATOMY OF PERITONEAL CAVITY

Abdominal cavity is the largest cavity. It encloses the peritoneal cavity between its parietal and serosal layers. Parietal layer clings to the wall of the parieties while visceral layer is intimately adherent to the viscera concerned. So their vascular supply and nerve supply are same as the parieties and the viscera respectively.

There are very lengthy organs in the peritoneal cavity. These had to be disciplined with limited, movements for proper functioning of the gut in particular and the body. In general, infections involving the parietal peritoneum impart protective 'board- like rigidity' to the abdominal wall. Referred pain from the viscera to a distant area is due to somatic and sympathetic nerves reaching the same spinal segment,

The abdominal cavity is an extensive space which extends upwards, deep to the costal margin, into the concavity of the diaphragm; and projects downwards and backwards into the bony pelvis as the pelvic cavity. Thus a considerable part of the abdominal

cavity is overlapped by the thoracic cage above, and the bony pelvis below.

NINE REGIONS OF THE ABDOMEN

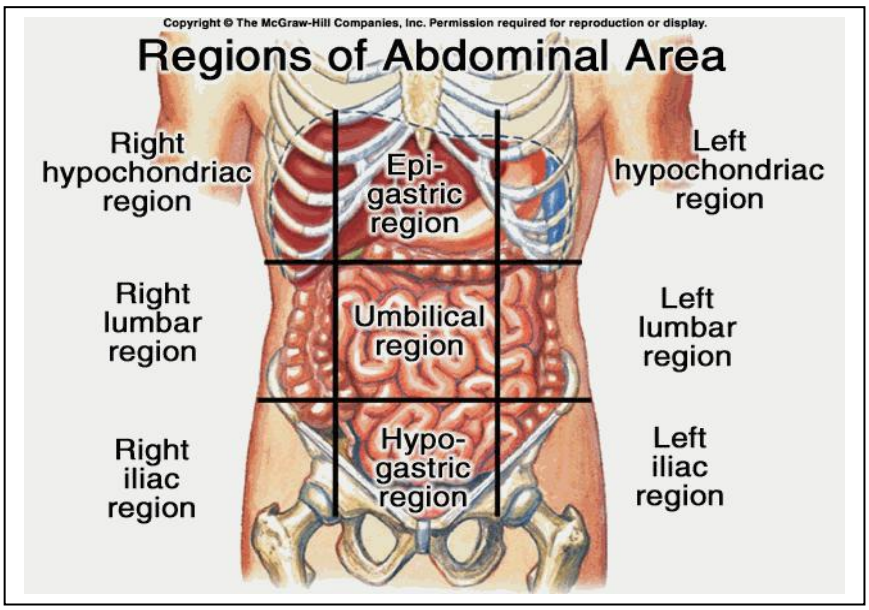
For the purpose of describing the location of viscera, the abdomen is divided into nine regions by four imaginary planes, two horizontal and two vertical. The horizontal planes are the transpyloric and transtubercular planes. The vertical planes are the right lateral and the left lateral planes.

Sometimes, another horizontal plane, the subcostal, is used instead of the transpyloric plane.

The transpyloric plane of Addison passes midway between the suprasternal notch and the pubic symphysis. It lies roughly a hand's breadth below the xiphisternal joint. Anteriorly, it passes through the tips of the ninth costal cartilage; and posteriorly through the body of vertebra L1 near its lower border.

The subcostal plane is sometimes used instead of the transpyloric plane. It passes through the lower borders of the 10^{th} costal cartilages, and the body of vertebra L3 near its upper border.

ABDOMINAL QUADRANTS



The transtubercular plane passes through the tubercles of the iliac crest and the body of vertebra L5 near its upper border.

The right and left lateral planes correspond to the midclavicular or mammary lines. Each of these vertical planes passes through the midinguinal point and crosses the tip of the ninth costal cartilage.

The nine regions marked out in this way are in three vertical zones, median, right and left. From above downwards, the median regions are epigastric, umbilical and hypogastric. The right and left regions, in the same order, are hypochondriac, lumbar and iliac.

PERITONEUM

Introduction

The peritoneum is a large serous membrane lining the abdominal cavity. Histologically, it is composed of an outer layer of fibrous tissue, which gives strength to the membrane and an inner layer of mesothelial cells which secrete a serous fluid which lubricates the surface, thus allowing free movements of viscera.

The peritoneum is in the form of a closed sac which is invaginated by a number of viscera. As a result the peritoneum is

divided into: (1) An outer or parietal layer; (2) an inner or visceral layer; and folds of peritoneum by which the viscera are suspended. The peritoneum which is a simple cavity, before being invaginated by viscera becomes highly complicated.

Parietal Peritoneum

1. It lines the inner surface of the abdominal and pelvic walls and the lower surface of the diaphragm. It is loosely attached to the walls by extraperitoneal connective tissue and can, therefore, be easily stripped.

2. Embryologically, it is derived from the somatic layer of the lateral plate mesoderm.

3. Its blood supply and nerve supply are the same as those of the overlying body wall. Because of the somatic innervation, parietal peritoneum is pain sensitive.

Visceral Peritoneum

1. It lines the outer surface of the viscera, to which it is adherent and cannot be stripped. In fact it forms a part and parcel of the viscera.

2. Embryologically, it is derived from the splanchnopleural layer of the lateral plate mesoderm.

3. Its blood supply and nerve supply are the same as those of the underlying viscera. Because of the' autonomic 'innervation, visceral peritoneum evokes pain when viscera is stretched, ischaemic or dis tended.

Peritoneal Cavity :

The viscera which invaginate the peritoneal cavity completely fill it so that the cavity is reduced to a potential space separating adjacent layers of peritoneum. Between these layers there is a thin film of serous fluid secreted by the mesothelial cells. This fluid performs a lubricating function and allow free movement of one peritoneal surface over another. Under abnormal circumstances there may be collection of fluid called ascites, or of blood called haemoperitoneum, or of air called pneumoperitoneum within the peritoneal cavity.

2. The peritoneal cavity is divided broadly into two parts. The main, larger part is known as the greater sac, and the smaller part, situated behind the stomach, the lesser omentum and the liver, is known 'as the lesser sac. The two sacs communicate with each other through

the epiploic foramen or foramen of Winslow or opening into the lesser sac.

3. Small pockets or recesses of the peritoneal cavity may be separated from the main cavity by small folds of peritoneum. These peritoneal recesses or fossae are of clinical importance. Internal hernia may take place into these recesses.

Sex Differences:

In the male, the peritoneum is a closed sac lined by mesothelium or flattened & epithelium. The female peritoneum hàs the following distinguishing features.

1. The peritoneal cavity communicates with the exterior through the uterine tubes.

2. The peritoneum covering the ovaries is lined by cubical epithelium.

3. The peritoneum covering the fimbria is lined by columnar ciliated epithelium.

Functions of Peritoneum

1. Movements of viscera

The chief function of the peritoneum is to provide a slippery surface for free movements of abdominal viscera. This permits peristaltic movements of the stomach and intestine, abdominal movements during respiration and periodic changes in the capacity of hollow viscera associated with their filling and evacuation. The efficiency of the intestines is greatly increased as a result of the wide range of mobility that is possible because the intestines are suspended by large folds of peritoneum.

2. Protection of viscera :

The peritoneum contains various cells which guard against infection. Lymphocytes present in normal peritoneal fluid provide both cellular and humoral Immunological defence mechanisms. The greater omentum has the power to move towards sites of infection and to seal them thus preventing spread of infection. For this reason the greater omentum is often designated as the “policeman of the abdomen”.

3. Absorption and dialysis : The mesothelium acts as a semipermeable membrane across which fluids and small molecules of various solutes can pass. Thus, the peritoneum can absorb fluid effusions from the peritoneal cavity. Water and crystalloids are absorbed directly into the blood capillaries, whereas colloids pass into lymphatics with the aid of phagocytes. The greater absorptive power of the upper abdomen or subphrenic area is due to its larger surface area and because movements aid absorption.

Therapeutically, considerable volumes of fluid can be administered through the peritoneal route. Conversely, metabolites, like urea, can be removed from the blood by artificially circulating fluid through the peritoneal cavity. This procedure is called peritoneal dialysis.

4. Healing power and adhesions : The cells of the peritoneum can transform into fibroblasts which promote healing, of wounds. They may also form abnormal adhesions causing obstruction in hollow organs.

5. Storage of fat: Peritoneal folds are capable of storing large amounts of fat, particularly in obese persons.

ANATOMY AND HISTOLOGY OF ILEUM

The jejunum and ileum are suspended from the posterior abdominal wall by the mesentery. Thèrefore they enjoy considerable mobility. The jejunum constitutes the upper two fifths of the mobile part of the small intestine, while the ileum constitutes the lower three fifths. The jejunum begins at the duodenojejunal flexure. The ileum terminates at the ileocaecal junction. The structure and functions of the jejunum and ileum correspond to the general description of the small intestine.

Root of Mesentery

It is marked by two parallel lines close ether, extending from the duodenojejunal flexure to the junction of the right lateral and transtubercular planes. The duodenojejunal flexure lies below the trans plane and 3 cm to the left of the median plane.

Blood supply :

The jejunum and ileum are supplied by branches from the superior mesenteric artery and are drained by corresponding veins.

Lymphatic Drainage :

Lymph from lacteals drains into plexuses in the wall of the gut. From there it passes into lymphatic vessels in the mesentery. Passing through numerous lymph nodes present in the mesentery and along the superior mesenteric artery, it ultimately drains into nodes present in front of the aorta at the origin of the superior mesenteric artery.

Ileum :

The villi are few, thin and finger like. Collection of lymphocytes in the forth of Peyer's patches in lamina propria extending into the submucosa is a characteristic feature.

PATHOPHYSIOLOGY OF PERFORATION

Perforative Peritonitis

The clinical picture is described in three stages.

(I) The first stage is known as peritonism i.e. It is due to leakage of bile into the peritoneal cavity (chemical peritonitis). This stage usually lasts for about six hours. Usually an adult male, who gives a previous history of fever, is suddenly seized with acute burning pain over the epigastrium. The pain may be referred to the tip of the right

shoulder due to irritation of the surface of the diaphragm. The pain may gradually gravitate down along the paracolic gutter to the right iliac fossa. At this stage one may misunderstand the pain to be due to acute appendicitis. The patient may or may not vomit. On examination there is high change in the pulse, respiration and temperature. Tenderness and muscle guard are constantly present over the site of upper half of the right rectus muscle. Great importance should be given on the diagnosis of this condition, at this stage survival of the patient gradually declines with passage of time. Diagnosis in the first stage mainly rests on two features viz onset of pain with a dramatic suddenness in a patient who has given a previous history of fever and muscle guard over the upper half of the right rectus muscle. In the late phase of this stage pain may be felt on the right iliac fossa which adds confusion to this diagnosis.

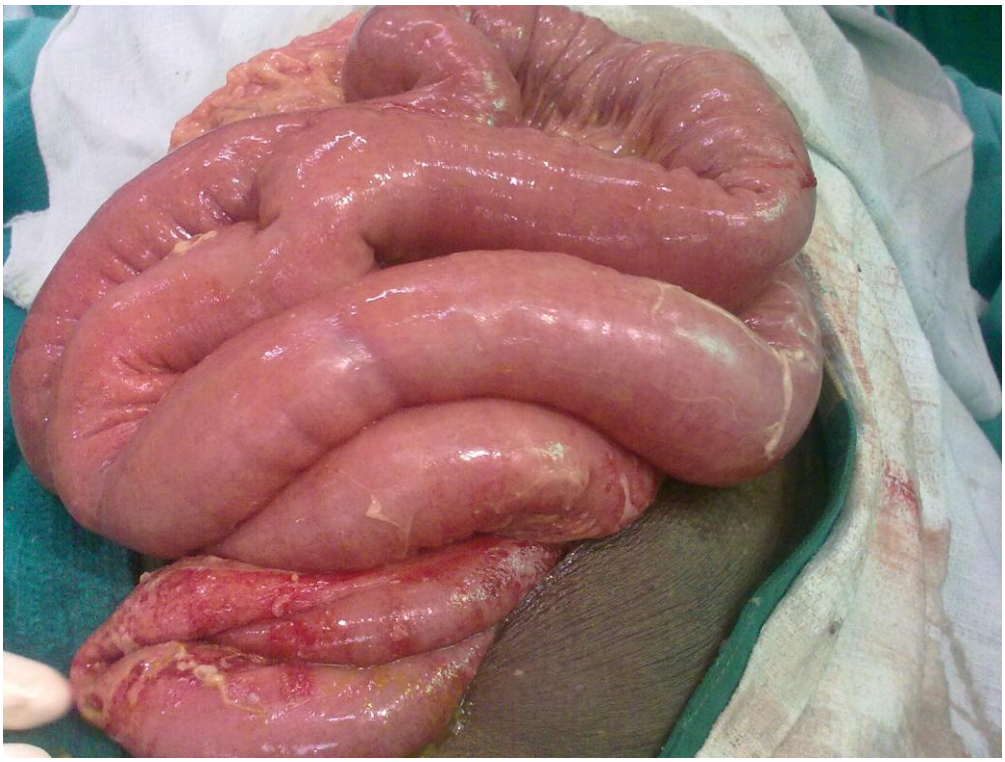
II) The second stage is known as the stage of reaction. The irritant fluid becomes diluted with the peritoneal exudate. The patient feels comfortable and nothing is more deplorab that the attending doctor sharing the patient's comfort. Symptoms are no doubt relieved but the signs are there and should be looked. Muscular rigidity continues to be present. The other two new features are obliteration

of liver dullness and shifting dullness. Rectal examination may elicit tenderness in the recto-vesical or recto-uterine pouch. Straight X-ray in sitting position will show air under the diaphragm in 70% of cases.

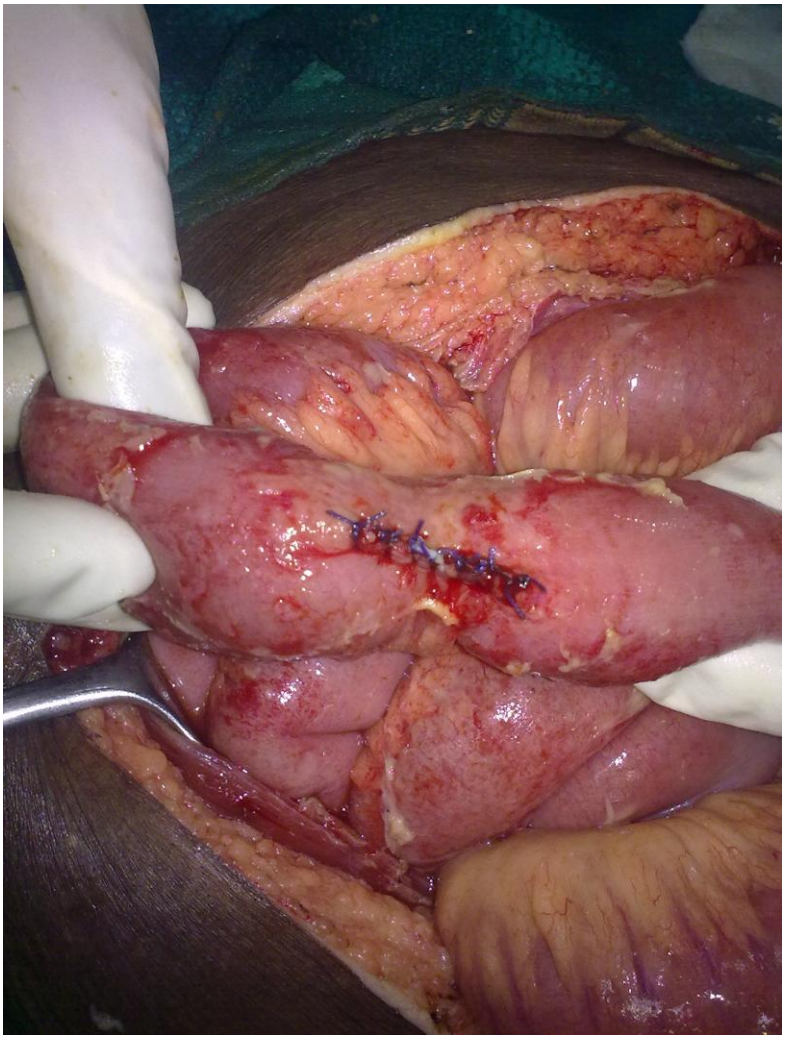
(III) The third or the final stage is the stage of diffuse peritonitis - and it indicates that the patient has gone a step towards the grave. The pinched and anxious face, sunken eyes and hollow cheeks — the so called facies hippocratica, with rising pulse rate which is low in volume and tension, persistent vomiting, 'board-like' rigidity of the abdomen, increasing distension of the abdomen all give hint to the diagnosis of this condition and imminent death.

ILEAL PERFORATION

PERFORATION IN THE ANTIMESENTERIC BORDER



PRIMARY CLOSURE OF ILEAL PERFORATION



METHODOLOGY

This study consists of patients admitted from May 2010 to October 2012. 40 patients of ileal perforation admitted to Govt. Rajaji Hospitals during this period were included in the study. A study of clinical features, investigations, operative procedures performed, postoperative morbidity and mortality and outcome was done. Jejunal, caecal, appendicular, gastric or duodenal perforations were excluded from the study. Traumatic ileal perforations were also excluded.

History with special reference to presence of fever, pain and abdominal distension, prior to admission was taken. Vital signs, hydration, abdominal distension, tenderness, guarding and presence of free fluid were noted. Systemic examination of cardiovascular, respiratory and central nervous system was done.

The following investigations were done as a routine

1. Hemoglobin
2. Bleeding and Clotting times
3. Blood sugar and urea and Serum creatinine

4. Chest X-Ray

5. Electrocardiogram

6. Peritoneal fluid culture

7. Pus culture in case of wound infection

In patients where in a resection was done the specimen was histopathologically examined. In all non-traumatic perforations the following additional investigations were done

1. Widal test
2. Blood Culture

All patients were resuscitated preoperatively with intravenous fluids and antibiotics. Patients unfit for surgery were initially treated with flank drains under local anaesthesia as a temporary measure prior to definitive laparotomy. Most cases received cefotaxime or ciprofloxacin with metronidazole. In case of gross peritoneal contamination aminoglycosides were added.

All patients underwent laparotomy under general anaesthesia. The amount and type of peritoneal contamination, number, site and size of perforations and procedure employed were noted. The following procedures were employed.

1. Simple two layer closure
2. Ileostomy
3. Resection and anastomosis
4. Ileo transverse anastomosis

For both closure and anastomosis, the inner all-coats layer was performed with 3-0 vicryl and the outer layer with silk.

Antibiotics were routinely given for 5-7 days unless the diagnosis was typhoid in which case antibiotics were continued for up to 10 days.

A diagnosis of typhoid was made only if Widal test was positive, or Salmonellae were isolated from blood or urine and if histopathological evidence of typhoid perforation was found. When the etiology of a non-traumatic perforation was not found, it was termed non-specific. Postoperative complications were noted. The factors influencing mortality and morbidity and outcome were assessed.

The various parameters were recorded in a proforma and tabulated.

RESULTS

Forty patients of ileal Perforation admitted between May 2010 and October 2012 were included in this study. Patients have been grouped into etiological categories, namely, typhoid, non-specific, and miscellaneous.

Etiology

The commonest cause of ileal perforation was typhoid. 13 patients had non specific perforations. One patient was diagnosed to have HIV with ileal perforation. The distribution is shown in table 1.

Table 1.

Table 1: Etiology of Ileal Perforation

Diagnosis	Frequency	Percentage
Typhoid	26	65
Nonspecific	13	32.5
HIV	1	2.5
Total	40	100

Table – 1

Age Distribution

Age in years	No.of cases	Percentage
< 20	4	10
21 – 40	20	50
41 – 60	14	35
> 60	2	5
Total	40	100

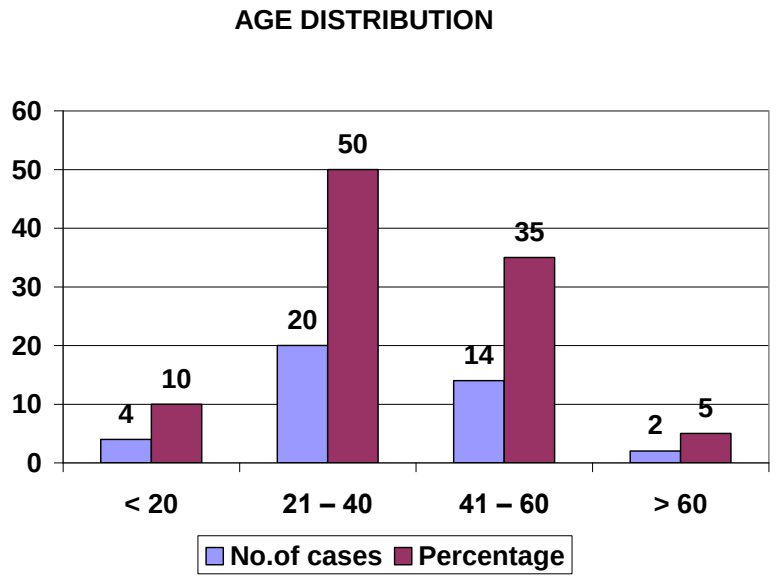


Table – 2

Sex Distribution

Sex	No.of cases	Percentage
Male	30	75
Female	10	25
Total	40	100

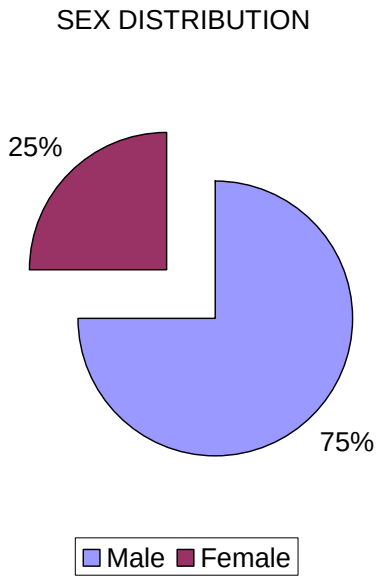


Table – 3

History of Fever

Fever	No.of cases	Percentage
< 4 days	10	25
5 – 10 days	27	67.5
> 10 days	3	7.5
Total	40	100

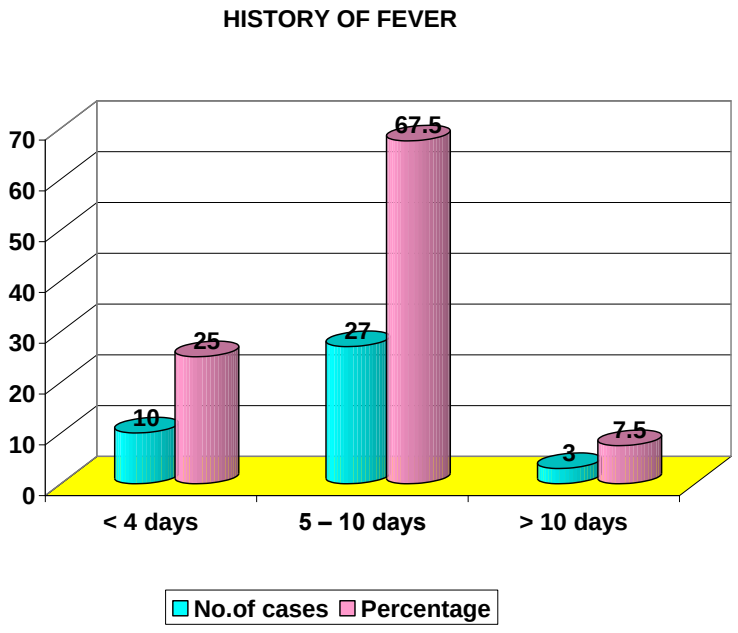


Table – 4

H/o Pain Abdomen

H/o Pain Abdomen in days	No.of cases	Percentage
< 2	22	55
3 - 4	14	35
5 - 6	4	10
Total	40	100

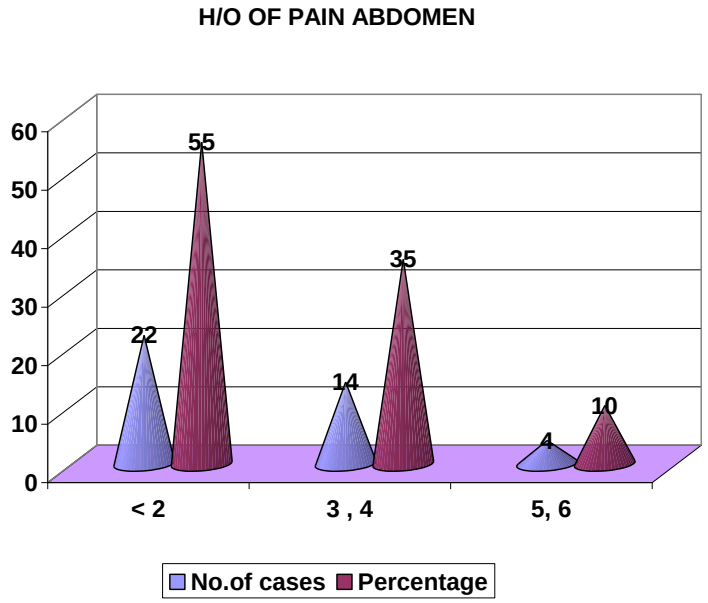


Table – 5

Site

Site in cms	No.of cases	Percentage
< 20	14	35
20 – 40	24	60
41 – 60	2	5
Total	40	100

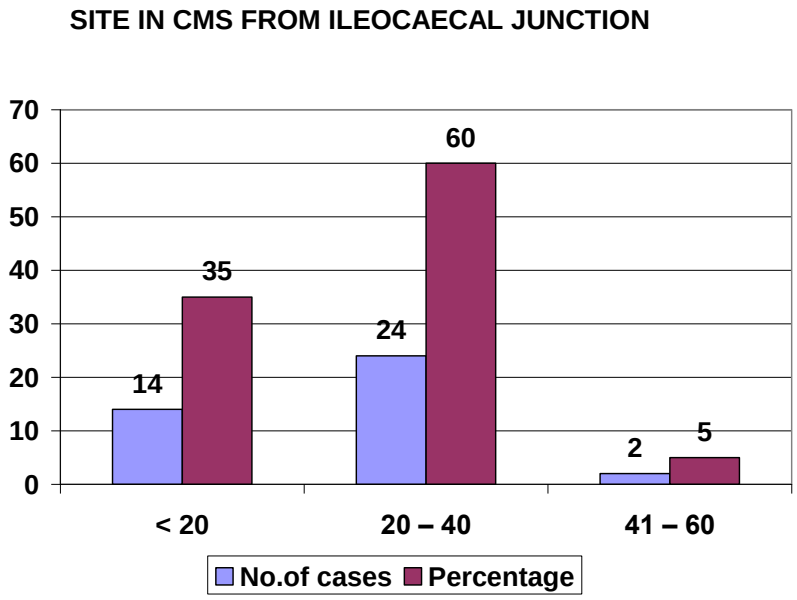


Table – 6

Number of Perforations

Number	No.of cases	Percentage
1	36	90
2	3	7.5
3	1	2.5
Total	40	100

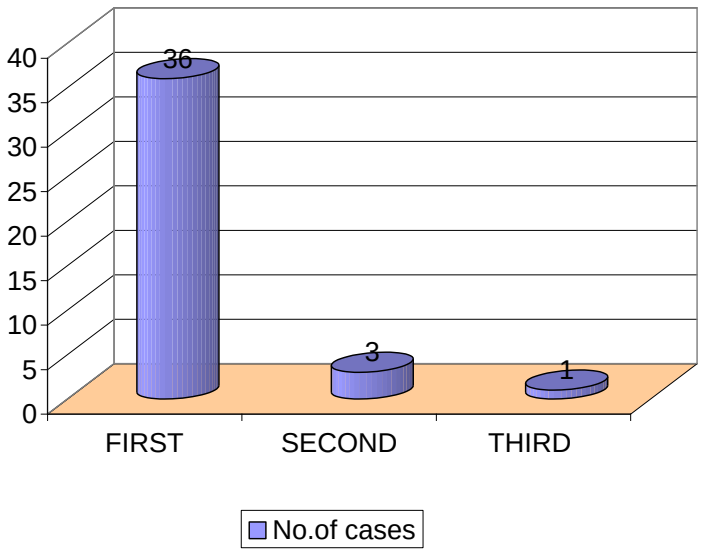


Table – 7

Management

Management	No.of cases	Percentage
Primary closure	31	77.5
Ileostomy	5	12.5
Ileo transverse anastomosis	3	7.5
Resection Anastomosis	1	2.5
Total	40	100

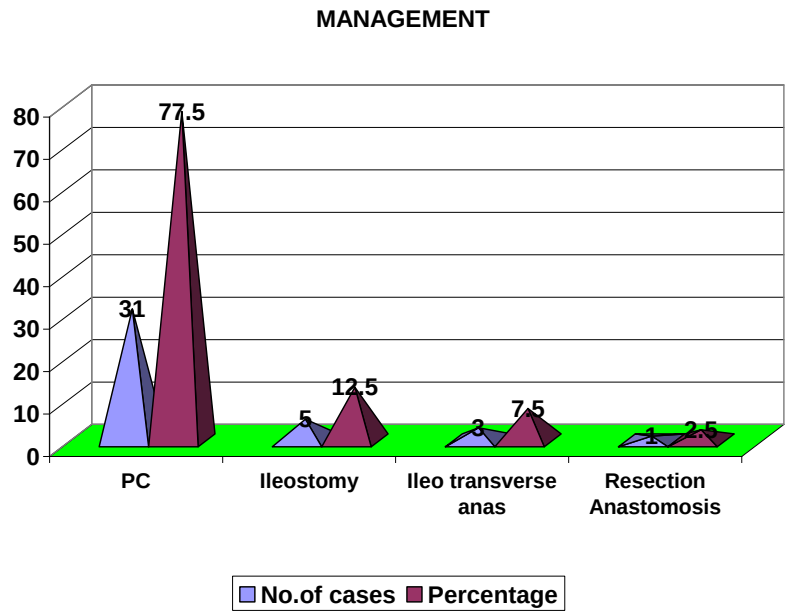


Table – 8

Widal

Blood culture	No.of cases	Percentage
Positive	26	65
Non Specific	14	35
Total	40	100

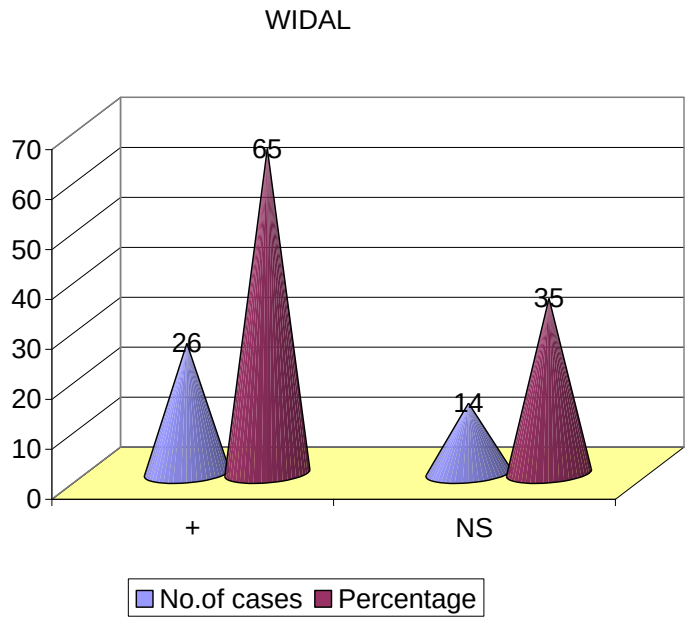


Table – 9

Secondary suturing

Suture	No.of cases	Percentage
Done	2	5
Nil	38	95
Total	40	100

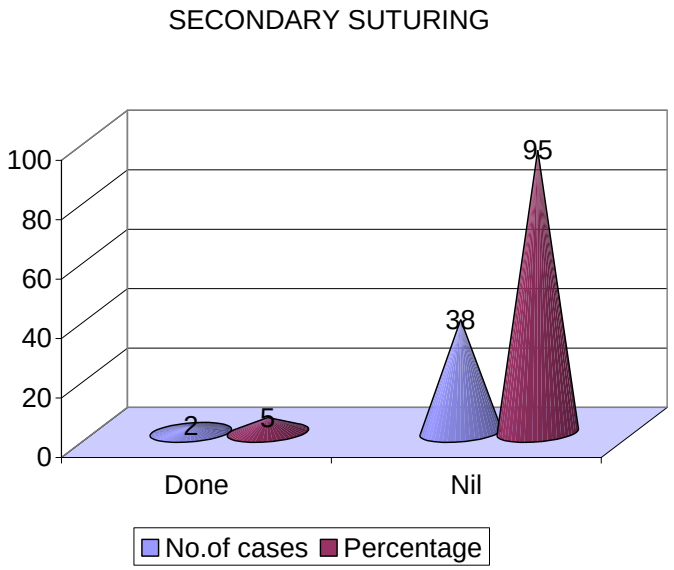
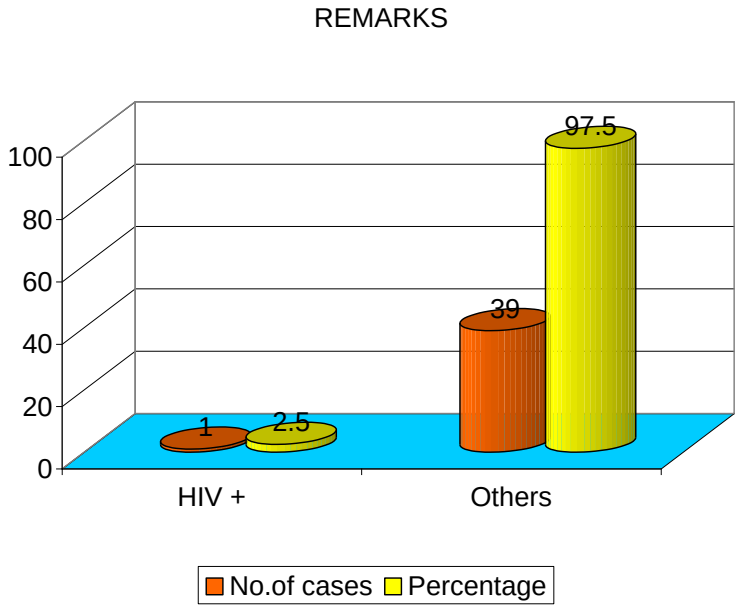


Table – 10

Remarks

Remarks	No.of cases	Percentage
HIV +	1	2.5
Others	39	97.5
Total	40	100



Age and Sex Incidence

The age of patients ranged from 15 to 84. Perforation commonly occurred in the second and third decade of life with 60% of patients between the ages of 20 and 40. The male to female ratio was 3:1.

Typhoid perforation commonly occurred in the second and third decades 62% of patients between the age of 20 and 40.The male to female ratio was 3:1.

Typhoid perforation commonly occurred in the second and third decades with 62% of cases in a similar age group.

The distributions of age and sex in all cases and etiology specific distributions are shown in tables 2 and 3.

Symptoms and Signs

Most of the patients presented with symptoms and signs of peritonitis. The commonest symptoms were abdominal pain, fever and vomiting. The commonest signs were abdominal tenderness, guarding, intra-abdominal free fluid and dehydration. Most patients of typhoid gave a history of fever. 12 % of patients were in shock.

Investigations

X-Ray:

Pneumoperitoneum in chest and erect abdominal x-ray was seen in 78% of patients. Features of intestinal obstruction, including dilated bowel loops with air-fluid levels in erect abdominal x-ray were seen 24% of patients.

Hematology and Biochemistry:

Hemoglobin was less that 8g/dL in 20(50%) of patients and Albumin of<3.5g/dL was seen in 10(25%) of cases. Azotemia as defined as a Blood urea of >52 mg/dL and/or Serum Creatinine > 2 mg/dL was seen in 40% of patients.

Microbiology: Blood cultures were done in 40 patients and growth was obtained in 4. Salmonella typhi was grown in 4 patients. The typhoid growths were sensitive to ciprofloxacin, cefotaxime, ceftriaxone and amikacin. Widal test was positive in 26 patients out of 40 tested.

Histopathology:

Pathological examination of either resected specimens of scrapings from the edge of the ulcer was done in 40 patients. All showed features of non-specific inflammation with no conclusive diagnosis.

Surgical Procedures

Simple 2-layer closure was the commonest procedure done (77.5%), end ileostomy (12.5%), ileo transverse anastomosis (7.5%) and resection and anastomosis in(2.5%) of patients.

Number and Site of Perforation

Multiple perforations occurred in 20% of patients, mostly in typhoid. Over 90% of perforations were within 2 feet (60 cms) from the ileocaecal junction and 62% within 30 cms.

Complications

Complications occurred in 4 (10%) of all cases (Tables 9, 10 and 11). The common complications seen were wound dehiscence.

The highest complication rate was seen with simple closure and the least with resection and anastomosis though this difference was not statistically significant.

Mortality

The mortality rate was 5%. The difference in mortality was not found to be statistically significant, septicemia and respiratory complications were the commonest causes of death.

Prognostic factors

Surgical Procedure

The type of surgical procedure did not influence the mortality or morbidity in ileal perforations and also in etiology specific analysis. In patients with typhoid, simplè closure had the highest mortality and resection anastomosis the highest complication rate but not statistically significant. In non-specific perforations simple closure had the highest mortality and complication rate but again not found to be significant.

DISCUSSION

The commonest cause of ileal perforation in the series was typhoid fever accounting for 65% of cases. Typhoid fever was the commonest cause of ileal perforation in tropical countries.

When the etiology of the perforation was not identified it was termed non-specific perforation. Non-specific perfóration was the second commonest cause in this study accounting for 32.5% of cases. Seven patients of non-specific perforation had fever prior to onset of abdominal symptoms. Widal test, blood culture and Histopathology were not suggestive of typhoid. These cases may be undiagnosed cases of typhoid.

There was a male preponderance with the male: female ratio in this study being This preponderance was seen in typhoid, non-specific

Most patients presented with features suggestive of peritonitis. Patients of both typhoid and non-specific perforations had similar presentation with respect to abdominal symptoms and signs. Patients with typhoid perforation had fever, abdominal pain and vomiting.

Examination revealed tenderness, guarding, distension and intraperitoneal free fluid. 6 patients were in shock on admission.

Chest X-ray is a useful investigation to detect hollow viscus perforation. Free gas was seen under the diaphragm in 78% of perforations and in 75% of typhoid perforation. Abdominal X-ray revealed gas of features suggestive of ileus. Pneumoperitoneum has been reported in 52% to 82% in studies by Hadley, Archampong, Tacyildiz and Vaidyanathan [17,19,22,48].

Widal was positive in 55% of tested cases and in 91% of patients of typhoid perforation. Widal was reported positive in 30% of patients with typhoid perforation by Kaul and in 46.1% of patients by Santillana [18,32] .It was reported positive in 75.5% of cases by Jarrett and in 73% by Vaidyanathan [42,48]. Four-fold increase in titres is considered more significant [34].

Salmonella typhi was grown in 4 (10% of tested) patients with ileal perforation in whom blood cultures were done. All cultures were sensitive to ciprofloxacin, cefotaxime and ceftriaxone. Hadley reported positive cultures in 22.2% and Santillana in 48% of patients [17,18] .Prior antibiotic therapy was probably responsible for the low

isolation the study [17,19,34] .Another cause may be delay in plating the samples.

In this study most patients of confirmed typhoid were treated with ciprofloxacin and metronidazole. The rest had a third generation cephalosporin (cefotaxime) and metronidazole.

In the management of typhoid perforation some authors advocated conservative management [36,37,38] .Presently there is no such controversy in the treatment of typhoid perforation with the current recommendation being surgical management [20].The various methods in use are local drains, simple closure, closure with omental patch, wedge resection, resection and anastomosis, ileotransverse anastomosis and ileostomy[25,28,32,40,4142] .In this study patients underwent simple closure, omental patch repair or resection anastomosis. No patients were treated by conservative measures, wedge resection, ileotransverse anastomosis or ileostomy. Resection was employed in typhoid or traumatic perforations wherein multiple perforations were found on laparotomy.

The mortality in this series was 5%. Typhoid perforations in this study thus showed a poorer prognosis than the other etiologies.

The surgical procedure did not influence either the morbidity or the mortality in patients irrespective of etiology. Simple closure was found to have a higher complication rate but this was not statistically significant. Eggleston reported that the procedure done did not influence outcome [26]. Talwar and Sharma reported that mortality was least with early primary closure and Anieh et al found mortality was highest with wedge resection and least with resection and anastomosis [41,42].

In patients of ileal perforation the significant factors influencing mortality are age greater than 50, female sex, feculent peritonitis, raised blood urea or creatinine as per the Manheim peritonitis index. In this study age greater than 50 and shock at presentation were significant factors influencing mortality. Trends were seen with fecal fistula formations, etiology of typhoid and preoperative azotemia. Sex, hemoglobin or albumin levels, number of perforations and type of peritoneal contamination were not found to be significant.

Archampong reported that urine output prior to surgery, blood urea and serum potassium affected survival in patients of typhoid perforation. Survival was independent of hemoglobin level, shock,

sickling status and number of perforations [19] .Mock reported that increasing number of perforations, generalised contamination of the peritoneal cavity and single layer closure influenced survival [21] .Eggleston in his series of 78 patients reported the shock, uremia, encephalopathy, fecal peritonitis and postoperative fecal fistula were predictors of mortality [26].

SUMMARY AND CONCLUSIONS

This study was conducted from May 2010 to October 2012. It includes forty cases of ileal perforation admitted to Govt. Rajaji Hospitals in that period. Etiology, presentation, management and outcome of patients with ileal perforations were studied with emphasis on typhoid, non-specific and traumatic perforations and the factors that influenced the prognosis.

- ✓ Typhoid is the most common cause of ileal perforation, followed by non-specific perforations.
- ✓ Patients have a male preponderance and are usually in the second and third decades of their lives.
- ✓ Widal serology is a useful test in the diagnosis of typhoid fever.
- ✓ Typhoid perforations have a significantly higher morbidity rate than non-specific perforations.
- ✓ Mortality in ileal perforations, especially typhoid is high, though the etiology is not a significant contributing factor

- ✓ The type of surgical procedure did not influence outcome, either morbidity or mortality.
- ✓ Morbidity was significantly influenced by age greater than 50, hypoalbuminemia and a diagnosis of typhoid as the cause of perforation.
- ✓ Mortality was significantly influenced by age greater than 50 and shock on admission.

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PROFORMA

Name	Age/Sex	IP No.
DoA	DoD	DoS
Stay		

History

Symptoms	**Present/Absent**	**Duration**
Abd Pain		
Fever		
Vomiting		
Constipation		
Loose stools		
Others		

Examination

PR	BP	RR
Temp	Hydration	Pallor
Jaundice	Others	

Per Abdomen

Distension	Liver Dullness	Guarding
Tenderness	Bowel Sounds	Free Fluid
Splenomegaly		

Other Systems

CVS	CNS
RS	OTHERS

Investigations

Hb	BI. Urea	S. Creat
S. Albumin	Electrolytes	
Chest X-Ray	Abdomen X-Ray	Others
4 Quadrant Aspiration	Peritoneal Fluid c/s	
Blood c/s	Widal	
HPE		

Diagnosis

Per øp Findings

Site	Number	Size
Size of Involved segment of ileum		Peritoneal fluid

Operative Details

Procedure Done	Suture Material
Duration of surgery	Closure

Post Op

Antibiotics	Duration
Blood Transfusion	Protein supplementation
Amino Acid Transfusion	Others

Complications and Treatment Given

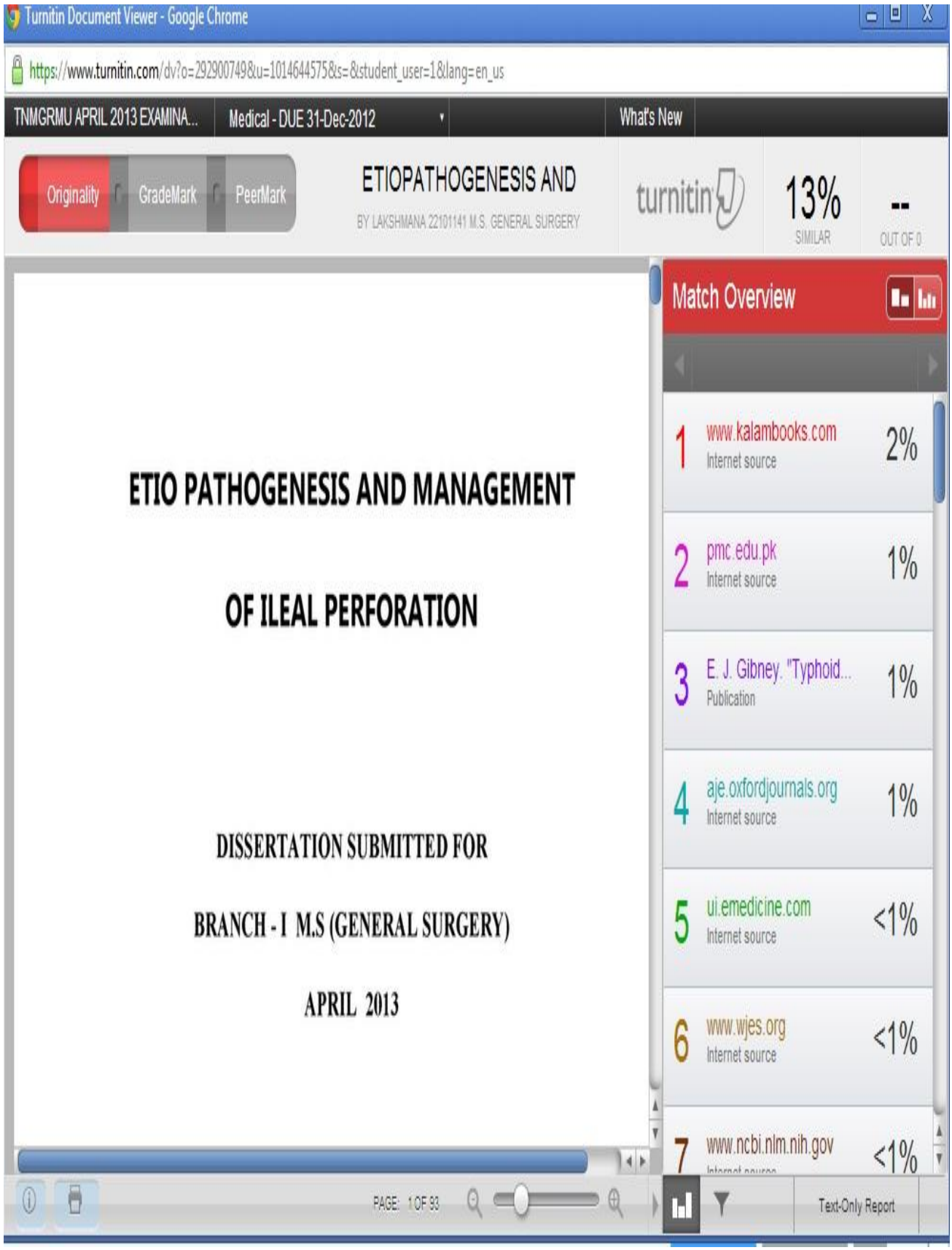
Postop Collection	Wound Infection	Wound dehiscence
Respiratory	Fecal fistula	Reoperation

Final Outcome

If Death, Time and Cause

MASTER CHART

S. No	Name	Age	Sex	IP No	H/O Pain	H/O Abominal istension	Site	Site	Number	Size	Mgmnt	Blood Culture	Wida	Post op	Secondary suturing	Remarks	Follow up
1	suresh	48	m	79678	8 d	2	20	20 cm	1	0.25x0.25	pc	+	+	burst abd	done	-	n
2	thiagaraja	47	m	63754	10 d	4	45	45 cm	1	0.5x0.5	pc	+	+	ue	-	-	n
3	chinnasamy	60	m	64512	10 d	3	12	12 cm	1	0.5x0.5	pc	+	+	ue	-	-	n
4	eswari	19	f	55019	10 d	4	25	25 cm	1	1x1	pc	+	+	ue	-	-	n
5	adhiraj	31	m	53268	12 d	6	28	20 cm ,28cm, 32cm	3	2x2 , 3x3, 1x1	ileostomy	ns	ns	death			
6	pandidurai	30	m	58413	10 d	4	15	15 cm	1	0.5x0.5	pc	+	+	ue	-	-	n
7	aarayee	60	f	65542	7 d	6	18	18 cm	1	1x1	pc	ns	ns	ue	-	-	n
8	chinnasamy	49	m	80973	6 d	4	20	20 cm	1	0.5x0.5	pc	+	+	ue	-	-	n
9	pitchaiammal	55	f	81942	5 d	2	25	25 cm	1	0.5x0.5	pc	ns	ns	ue	-	-	n
10	shanmugavalli	51	f	36302	4 d	4	30	30 cm	1	0.5x0.5	pc	ns	ns	ue	-	hiv +	n
11	periyasamy	55	m	47781	6 d	4	60	60 cm	1	0.5x0.5	pc	+	+	ue	-	-	n
12	thavasi	83	m	34793	10 d	6	40	40 cm	1	2x2	ileostomy	ns	ns	death			
13	muthukumar	17	m	34819	12 d	2	20	20 cm	1	0.25x0.25	pc	+	+	burst abd	done	-	n
14	palaniandi	39	m	49379	10 d	2	20	20 cm	1	1x1	pc	ns	ns	ue	-	-	n
15	alagar	37	m	75886	10 d	3	25	10cm, 25cm	2	1x1 , 0.5x0.5	ileo transverse anas	ns	ns	ue	-	-	n
16	nagaraj	23	m	13341	6 d	2	20	20 cm	2	1x1 , 0.5x0.5	ileostomy	ns	ns	ue	-	-	n
17	krishna	43	f	42981	10 d	3	18	18 cm	1	2x2	ileo transverse anas	ns	ns	ue	-	-	n
18	ramakrishnan	30	m	37890	10 d	1	10	10 cm	1	0.25x0.25	pc	+	+	ue	-	-	n
19	md kani	60	m	41047	15 d	2	16	16 cm	1	2x2.5	ileostomy	+	+	ue	-	-	n
20	chandran	31	m	58850	10 d	3	20	20 cm	1	0.5x0.5	pc	+	+	ue	-	-	n
21	alagar	37	m	58861	6 d	2	20	20 cm	1	0.25x0.25	pc	+	+	ue	-	-	n
22	nagurkani	65	m	12867	10 d	2	10	10 cm	1	0.25x0.25	pc	+	+	ue	-	-	n
23	irulan	18	m	19489	10 d	1	15	15 cm	1	0.25x0.25	pc	+	+	ue	-	-	n
24	nagendran	40	m	53633	8 d	6	20	20 cm	1	0.5x0.25	pc	+	+	ue	-	-	n
25	pitchaimuthu	23	f	64961	10 d	2	16	16 cm	1	0.25x0.25	pc	+	+	ue	-	-	n
26	umarani	43	f	66639	12 d	4	20	20 cm	2	0.5x0.5, 1x1	resection anastomosis	ns	ns	ue	-	-	n
27	thangam	35	m	80788	10 d	2	16	16 cm	1	1x1	ileo transverse anas	ns	ns	ue	-	-	n
28	raja	26	m	84174	10 d	1	10	10 cm	1	025x0.25	pc	+	+	ue	-	-	n
29	lakshmi	40	f	84192	16 d	2	12	12 cm	1	0.25x0.25	pc	+	+	ue	-	-	n
30	murthy	40	m	51716	10 d	1	16	16 cm	1	0.5x0.5	pc	ns	ns	ue	-	-	n
31	vellaiammal	24	f	51819	10 d	2	20	20 cm	1	0.25x0.25	pc	ns	ns	ue	-	-	n
32	sadayandi	47	m	22466	12 d	3	24	24 cm	1	0.5x0.5	pc	+	+	ue	-	-	n
33	satish kumar	25	m	31478	10 d	2	25	25 cm	1	0.25x0.25	pc	ns	ns	ue	-	-	n
34	kavita	22	f	31499	6 d	1	20	20 cm	1	0.25x0.25	pc	+	+	ue	-	-	n
35	raja	32	m	37819	4 d	1	20	20 cm	1	0.25x0.25	pc	+	+	ue	-	-	n
36	umarbasa	49	m	44595	10 d	3	30	30 cm	1	0.5x0.5	pc	+	+	ue	-	-	n
37	alagu	50	m	68933	12 d	4	36	36 cm	1	2x2	ileostomy	+	+	ue	-	-	n
38	ramanathan	40	m	30941	10 d	2	30	30 cm	1	0.5x0.5	pc	+	+	ue	-	-	n
39	manikandan	35	m	68466	12 d	2	20	20 cm	1	0.5x0.5	pc	+	+	ue	-	-	n
40	asaipandi	19	m	49544	14 d	2	16	16 cm	1	0.25x0.25	pc	+	+	ue	-	-	n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